CHANGES IN ACTIVITY OF THE URETHRAL SPHINCTER DURING STIMULATION OF MECHANORECEPTORS OF THE URINARY BLADDER

V. A. Kuz'menko

UDC 612.467.1-06:612.467.3

A steady increase in fluid pressure in the urinary bladder causes either an increase in bioelectrical activity of the urethral sphincter at low pressure values and inhibition at high values, or inhibition at high values, or inhibition at all values of intravesical pressure. Changes of the second type were observed in cases when the tone of the empty urinary bladder was raised at the beginning of stimulation.

* * *

In accordance with the classical views [5], an increase in fluid pressure in the urinary bladder up to a threshold causes relaxation of the urethral sphincter, which is formed from striated muscles. However, according to Lagutina [3, 4], the character of reaction of the sphincter to the creation of a constant intravesical pressure depends on its level: weak stimulation increases activity of the sphincter while strong stimulation inhibits its activity. Other investigators have observed a similar relationship [6, 7]. At the same time, it has been shown that with an increase in the total afferent flow along fibers of the pudendal nerves, activity of the sphincter at first shows a reflex increase and then falls sharply, ultimately disappearing altogether [2].

In this investigation changes in electrical activity of muscles of the urethral sphincter were studied during a steady increase in intravesical pressure and also during simultaneous stimulation of receptors of the urinary bladder and the urethral sphincter.

EXPERIMENTAL METHOD

A series of 40 acute experiments was carried out on adult cats under urethane anesthesia (1 g/kg intravenously). A cannula was tied into the apex of the bladder, through which warm physiological saline was introduced, and the intravesical pressure measured by means of an electromanometer. The bladder neck was clamped. Receptors of the sphincter were stimulated by passing physiological saline through the urethra. The intravesical pressure, bioelectrical activity of the branch of the pelvic nerve and muscles of the urethral sphincter, and integrated values of this activity (time constant of integration 0.5 sec), and time marker were recorded simultaneously on a loop oscillograph.

EXPERIMENTAL RESULTS

In most experiments (83%), when the bladder was empty, electrical activity of muscles of the urethral sphincter of varied degree was observed. In these experiments a steady increase in intravesical pressure caused definite changes in activity. In a small proportion of experiments (17%) the initial activity was below the noise level of the amplifier (3-4 μV) and remained unchanged as the intravesical pressure was raised from 0 to 80 mm Hg, 5 times higher than the mean pressure at which micturition begins in anesthetized cats.

In 33% of experiments a steady increase in intravesical pressure caused only inhibition of the electrical activity of the sphincter. In the remaining 50% of experiments, a small increase in pressure (on the average to 6.2 mm Hg) increased activity, while a further increase in pressure caused its inhibition (Fig. 1). Mean values of initial activity of the sphincter and branch of the pelvic nerve (Table 1) were determined for each group of experiments (Table 1), and curves showing changes in the mean value of sphincter activity during an increase in intravesical pressure were plotted (Fig. 2A and B).

M. F. Vladimirskii Moscow Regional Clinical Research Institute. (Presented by Academician V. N. Chernigovskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 67, No. 6, pp. 21-24, June, 1969. Original article submitted November 1, 1968.

Fig. 1. Reflex changes in electrical activity of muscles of urethral sphincter in response to steady increase in intravesical pressure. From top to bottom: intravesical pressure; bioelectrical activity of pelvic nerve and its integral; bioelectrical activity of muscles of urethral sphincter and its integral; time marker, 1 sec.

TABLE 1. Character of Reflex Changes in Activity of Urethral Sphincter in Response to Steady Increase in Intravesical Pressure

Group no.	Character of change in sphincter activity	No. of expts.	Relative percentage	Mean value of initial sphincter activity (in conventional units)	Mean value of initial activity in pelvic nerves (in conventional units)
1	Increase changing to inhibition	20	50	0.43	0.2
2	Inhibition	13	33	0.77	0.58
3	No change	7	17	Less than 0.08	0.16

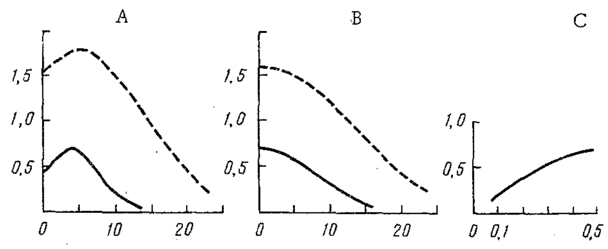

Fig. 2. Changes in mean activity of urethral sphincter (ordinate) in response to steady increase in intravesical pressure. A) Increase changing into inhibition; B) inhibition. Continuous line shows change in background activity, broken line, change in activity during simultaneous stimulation of receptors of sphincter and bladder; C) relationship between mean activity of sphincter and impulse activity in pelvic nerve from the empty urinary bladder. Ordinate for all curves: integral of activity relative to integral of calibration signal of 20 μV, 400 Hz. Abscissa: in C (these same units; in A and B) intravesical pressure (in mm Hg).

Comparison of the curves in Fig. 2A and B shows that in the animals of group 2 the initial sphincter activity was increased and was approximately equal to the maximum reached during reflex increase in activity in the animals of group 1. Meanwhile, in the animals of group 2, the initial bioelectrical activity in the pelvic nerves was increased (Table 1). Comparison of the results for different animals of group 1 shows that with a greater increase in initial activity of the pelvic nerves, on the average higher initial activity of the sphincter was observed (Fig. 2C), and the maximal increase in this activity in response to weak stimulation of the vesical receptors was lower in value and occurred at a lower pressure. These facts suggest that the uniform change in activity observed in the animals of group 2 was a special case of a more general biphasic change observed in the cats of group 1. The transition from one type of response to the other occurs in cases when sphincter activity before the beginning of stimulation of the urinary bladder is increased by a reflex mechanism in response to an increase in tone of its smooth muscle. In some experiments, during repeated distention of the bladder, responses characteristic of the different groups of animals were observed.

To determine the character of interaction between the intrinsic reflex of the sphincter (whose afferent and efferent pathways lie in the pudendal somatic nerves) and the reflex from the urinary bladder on the sphincter, in 20 experiments simultaneous stimulation of the two reflexogenic zones was applied: a steady increase in intravesical pressure against the background of the passage of fluid through the urethra. In these experiments stimulation of the sphincter receptors when the bladder was empty led to an increase in bioelectrical activity of the muscles of the urethral sphincter. The reflex increase in activity was much greater than in experiments with stimulation of the vesical receptors, its mean value being 1.5 units. In 55% of experiments distention of the bladder while fluid was passed through the urethra caused inhibition of sphincter activity, and in 45% of experiments an increase in its activity at low pressures and inhibition at high pressures (Fig. 2A and B).

Similar results were obtained in experiments when stimulation of the mechanoreceptors of the urinary bladder was combined with stimulation of extero-receptors of the genital region and skin of the perineum.

In most experiments, with a steady increase in intravesical pressure, weak stimulation thus caused an increase in sphincter activity and some stimulation inhibited its activity. This result agrees with the observations of Lagutina [3, 4], who described a biphasic relationship between afferent impulse activity from the urinary bladder, on the one hand, and efferent activity of the pudendal nerves and bioelectrical activity of the sphincter muscles on the other. The change observed in some of the present experiments in activity to a monophasic type was due to a temporary increase in level of the initial activity of the pelvic nerves (Table 1, Fig. 2C). It will be recalled that a temporary increase in afferent impulse activity from the empty urinary bladder has frequently been observed [1, 5, 8].

The increase in sphincter activity in response to stimulation of the vesical mechanoreceptors was much weaker than during stimulation of the receptors of the sphincter. Experiments with simultaneous stimulation of the two reflexogenic zones showed that the mean increase in sphincter activity during a steady increase in intravesical pressure was practically the same whether the initial sphincter activity was low or high, if the increase in initial activity was not connected with stimulation of the vesical receptors. In some experiments, however, stimulation of the sphincter receptors evidently led to an increase in tone of the urinary bladder (Barrington's second reflex [5, 6]) and, as a result of this, to increased sphincter activity. This explains the fact that during simultaneous stimulation of the two reflexogenic zones, the relative number of monophasic inhibition responses was higher than during stimulation of the vesical receptors only.

The character of changes in sphincter activity in response to a steady increase in intravesical pressure did not change significantly with an increase in the afferent flow from the sphincter. It is, therefore, reasonable to conclude that interaction between the intrinsic sphincter reflex and the reflex from the urinary bladder on the sphincter demonstrate algebraic summation of their final effects.

LITERATURE CITED

1. N. A. Adamovich, Transactions of the I. P. Pavlov Institute of Physiology [in Russian], Vol. 3, Moscow–Leningrad (1954), p. 490.
2. V. A. Kuz'menko, Byull. Éksperim. Biol. i Med., No. 3, 6 (1969).
3. T. S. Lagutina, Byull. Éksperim. Biol. i Med., No. 7, 3 (1957).
4. T. S. Lagutina, Fiziol. Zh. SSSR, No. 2, 214 (1960).
5. O. S. Merkulova and A. S. Mirkin, Fiziol. Zh. SSSR, No. 6, 721 (1966).
6. J. P. Evans, J. Physiol. (London), 86, 396 (1936).
7. R. C. Garry, F. D. M. Roberts, and J. K. Todd, J. Physiol. (London), 149, 653 (1959).
8. M. Talaat, J. Physiol. (London), 89, 1 (1937).